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AURICULO-VENTRICULAR HEART-BLOCK AS A RESULT OF ASPHYXIA.*

By THOMAS LEWIS and G. C. MATHISON.

(*From University College Hospital Medical School.*)

In an article upon the mammalian spinal animal, recently published, Sherrington[2] speaks of a sudden diminution of heart rate in asphyxia. He writes as follows: "From a frequency of 300 per minute it now absolutely abruptly drops to 140 to 150 per minute," and again, he says, "This sudden check of the heart's rate is not due to vagus action, for it occurs still quite typically after administration of atropin has set aside all cardiac inhibition obtainable by stimulation of the vagus trunk in the neck."

This year Roaf and Sherrington[1], writing upon the same subject, state that "At a certain stage of asphyxia there ensues, as shown in the previous paper, an abrupt reduction in the frequency of the ventricular beat, not due to vagus inhibition. It is in fact due to heart-block."

In view of the fact that neither Sherrington nor Roaf and Sherrington have brought forward evidence of the occurrence of heart-block during asphyxia, and knowing that an apparent halving of ventricular rate may occur in the mammalian heart, in the absence of heart-block, and in the absence of vagal inhibition, we considered it advisable to investigate the matter more closely. Using Edelmann's large pattern of Einthoven's string galvanometer, we have taken electrocardiograms from the right fore limb and left hind limb of cats.

The heart contractions have been recorded in the decapitated or spinal cat, and in the decerebrate animal under artificial respiration. They have also been recorded in the intact and curarised animal, with the vagi intact and with the vagi divided. Further, they have been registered in the spinal animal under doses of atropine, sufficient to paralyse the vagus terminations, as tested by faradisation of this nerve in the neck. Our results, in so far as they are stated in this paper, have been uniform.

It was desirable that the records should be taken at a moderately fast rate for purposes of measurement, and as a continuous record of a complete asphyxial observation, from its start to its finish, was impossible at such speeds, we took sample tracings at intervals of about 30 sec.. These intervals, as stated in the accompanying table, are sometimes shorter and sometimes longer than 30 sec., for in watching the movements of the shadow

*An account of observations undertaken under the tenure of Beit Memorial Fellowships.

of the string, it is not difficult to appreciate certain changes in the mechanism of the heart-beat, and often with the onset of such a change we obtained an early or late tracing. The first table which is given is a fairly characteristic example of such an observation. It differs from the remaining experiments in the fact that the onset of heart-block is, as a rule, earlier than is here shown.

TABLE I. Cat 1. Obs. 4. Decapitated Cat. Artificial Ventilation. 1/100 gr. atropine sulphate.

Duration of Asphyxia in min. & sec..	Auricular Rate.	P-R. Interval.	Ventricular Rate.	REMARKS.
0,0	124	·08	124	
0,30	125	·08—·09	125	
1,0	127	·07—·08	127	
2,0	127	·08	127	
2,15	127	·08	127	
2,30	161	·08—·10	161	
3,15	180	·08	180	
3,30	182	·08	182	Sl. variation of auricular rate.
4,0	161	·08	161	
4,15	142	·12—·13	142	Prolongation of P-R interval.
5,0	122	·12—·13	122	Sl. variation of auricular rate.
5,30	113	·12—·13	113	
5,45	107	·12—·13	107	
6,0	97	·14—·16	48·5	2 : 1 H.B.. Sl. variation of auricular rate.
6,30	96	·16	48	
7,30	90	—	52	C. H. B.

With restoration of ventilation the heart rate quickened to the original rate, and the heart-block disappeared.

TABLE II. Cat 10. Obs. 4. Intact animal; artificial respiration; urethane and curare; vagi cut.

Duration of Asphyxia.	Auricular Rate.	P-R. Interval.	Ventricular Rate.	REMARKS.
0,0	192	·10	192	
1,0	192	·10	192	
1,15	187	·10 +	187	
1,30	187	·10	187	
1,45	205	·10	205	T increasing
1,50	205	·12	205	T markedly exaggerated
2,30	150	·13	150	
3,0	130	·14—·15	130	
3,5	126	·15—·16	63	2 : 1 H.B.
4,15	105	—	67	C.H.B. One extrasystole.
4,40	101	—	59	C.H.B.
5,10	98	·18	49	2 : 1.
5,45	—	—	33	Auricle not recording.

The heart subsequently showed complete recovery, the P-R interval measuring ·08—·10 sec (cf. Fig. 4c.).

From left to right it indicates the duration of asphyxia, the rate of the auricle per minute, the P-R intervals in seconds, and the ventricular rate.

The second table is similarly arranged ; the time relationships are more typical. It shows an exceptional occurrence, the break back of complete to 2 : 1 heart-block.*

Within two or three minutes of the onset of asphyxia the rate of the auricles and consequently that of the ventricles increases. At $1\frac{1}{2}$ to $4\frac{1}{4}$ minutes (usually early) a prolongation of the P-R interval occurs, and this is the first evidence of the subsequent fully developed heart-block. It is either accompanied or directly preceded by a retardation of the auricular rate, and the decrease may be considerable. Following upon this stage is a further and steady decrease of auricular rate, and it is associated with the appearance of other grades of heart-block, of which examples are published (Fig. 1). The prolongation of the P-R interval becomes exaggerated, and it may lengthen to double or treble the original measurement (the longest interval recorded has been 0·3 sec.). (The normal interval for cats is about 0·08 to 0·1 sec.). Within two minutes (usually less) of this evidence of diminished conductivity of impulse transmission from auricle to ventricle, auricular impulses fail to reach the ventricle. At first there may be an occasional dropped beat (Figs. 2 and 3), there may be dropped alternate beats (2 : 1 heart-block, Fig. 1 III). Where single beats are dropped a condition comparable to that which is found in clinical cases has been met with. The already lengthened P-R interval rapidly increases still further up to the point where the dropped beat is seen. This preliminary lengthening of the P-R interval is clearly shown in Figs. 2 and 3 and the intervals increase to such an extent that the auricular systoles (P) fall back upon the preceding ventricular systoles (R and T), so that the contraction is partially synchronous in the two chambers. The first P-R interval succeeding the failure of a ventricular response is invariably relatively short, a shortening which has been frequently pointed out in clinical cases and which is attributable to the long rest which precedes it.

Following upon the stage of prolonged P-R interval or upon a period of dropped beats, 2 : 1 heart-block manifests itself (Fig. 1 III). The auricular rate during this period is always markedly retarded (despite the section of the vagi, or the administration of atropine), and the P-R intervals retain their increased length. They may be shorter, or of the same length or even slightly longer than the longest P-R intervals of the preceding phase. They are usually long as in Fig. 1. The duration of the 2 : 1 block is variable, but generally continues for a minute or two. It is succeeded by a period of complete heart-block, in which the auricular rate is approximately twice that of the ventricle, but in which there is no simple arithmetical ratio between the rhythms of the two chambers. There is complete dissociation, and the auricular variations (P) fall at varying times in relationship to the ventricular

* This reappearance of 2 : 1 heart-block does not necessarily indicate a decrease of impairment of conductivity. It may be explained as a result of the slowing of an ideoventricular rhythm.

representatives (R and T). Frequently the representatives of auricle and ventricle coincide, a phenomenon well shown in Fig. 1 IV. The first P of this figure falls in the centre of the ventricular systole, the second in the middle of diastole, while the third coincides with and raises the apex of the variation T of the second ventricular systole. An auricular variation is known to have occurred at this instant, for the superimposition (comparing the T variations of the two ventricular cycles) is exact, and a P variation is expected at this point. We have a number of curves of complete heart-block produced in this way, but not so many examples as we have of the preliminary stages, for the condition of the animal becomes critical, and often re-establishment of respiration and recovery is desired. Nevertheless, recovery and absolute recovery of the conductive functions may occur subsequent to the period of complete dissociation, an event which we have thrice witnessed, and which is exemplified in Fig. 4 *c*.

The recovery from complete heart-block is relatively slow, and is pursued through similar stages to those of its production, though the order is reversed. Recovery from 2 : 1 heart-block is faster. There is a short latent interval of about 10 to 20 sec., and then within a few heart cycles the condition 2 : 1 heart-block passes to one of complete restoration ; that is to say, the P-R interval returns to its original length at the commencement of the observation (Fig. 4 *a* and *b*). The change is the more remarkable for its suddenness, in view of the acceleration of auricular rate which accompanies it (Fig. 4 *a* and *b*). Recovery is permanent when it is complete.

During the course of the experiments we have noted many other phenomena of interest, but these, being still obscure, we reserve—two further observations alone require description at the present time.

1). At or about the time when the heart appears to waver between a condition of 2 : 1 heart-block and regular sequential contraction accompanied by prolongation of the P-R interval, it not infrequently happens that passing into the latter state it exhibits a regular alternation of the P-R intervals. This in turn gives rise to a slight grade of ventricular bigeminy, for the long pause delays the onset of the ventricular systole for which it provides an impulse. We have no explanation to offer for the phenomenon ; it is illustrated in Fig. 5.

2). The ventricular rate may increase at the onset of complete heart-block. This acceleration demonstrates the relative or absolute inactivity of impulse formation in the ventricle during the preceding stage of 2 : 1 heart-block. The auricular rate meanwhile may remain constant or diminish ; it does not accelerate as does that of the ventricle.

Conclusions.

1. In the experimental animal, as a result of asphyxia, heart-block in the grades in which it is encountered in clinical cases, is of almost

Fig. 1. I-IV. × ½ linear. Cat. 1, observation 4. Four electrocardiograms from a single period of asphyxia. Taken from a decapitated and atropinised cat. They show varying grades of heart-block in the order of their onset, in the fourth observation upon this particular animal. The time signal is in 0·2 sec. in all figures. The P-R intervals are marked in sec..

I. Curve taken 1 min. after the onset of asphyxia. The P-R interval is 0·08 sec..

II. Curve taken 5 min. 30 sec. after onset. The P-R interval, which first showed increase 4 min. 15 sec. after onset, is 0·12 to 0·13 sec..

III. Curve taken 6 min. after onset. 2 : 1 heart-block is present. The P-R interval is 0·15 sec..

IV. Curve taken 7 min. 30 sec. after onset. Complete heart-block is present.

The heart subsequently recovered its original rate, and the heart-block disappeared with the restoration of respiration.

Fig. 2. × ⅔ linear. Cat 5, observation 3. A curve taken soon after the onset of asphyxia, from a decapitated cat. It shows two dropped beats and the lengthening of P-R interval which leads up to them and shortening of P-R interval succeeding them. Certain of the auricular systoles (P) fall back on the preceding ventricular systoles (R and P). The P-R intervals are marked in sec..

Fig. 3. × ⅔ linear. Cat 10, observation 2. Curve taken from an intact cat, 3 min. after onset of asphyxia ; vagi uncut, under urethane and curare. Showing dropped beats and the prolongation of P-R intervals leading up to them.

Fig. 4 *a*. × ⅔ linear. Cat 10, observation 3. From the same cat. Showing the rapid recovery from 2 : 1 heart-block. (Compare with succeeding figure.)

Fig. 4 *b*. × ⅔ linear. Cat 10, observation 3. A strip of curve of 4 sec. duration has been excised between curves 4 *a* and 4 *b*. The P-R interval is now 0·08 sec..

Fig. 4 *c*. × ⅔ linear. Cat 10, observation 7. Subsequent to the recovery shown in Fig. 4 *a* and *b* the vagi were cut and the cat underwent four further asphyxial periods, in two of which complete heart-block was developed. The curve is given to show the complete recovery of conduction succeeding this dissociation.

Fig. 5. × ½ linear. Cat 1. Alternation of the P-R interval. A strip of curve lying between two periods of 2 : 1 heart-block.

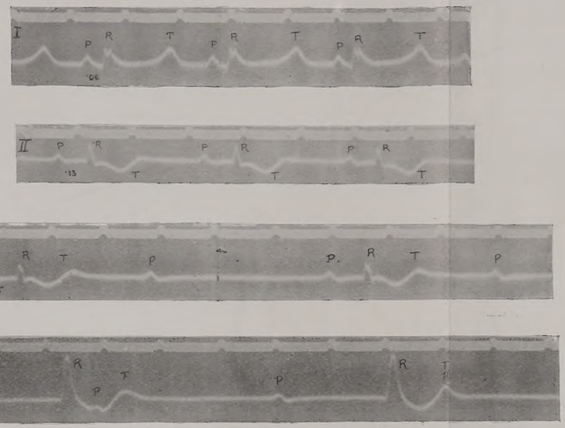

Fig. 1.

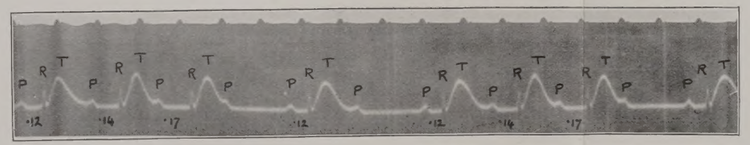

Fig. 2.

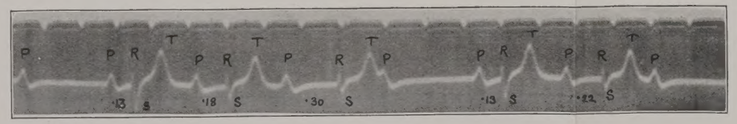

Fig. 3.

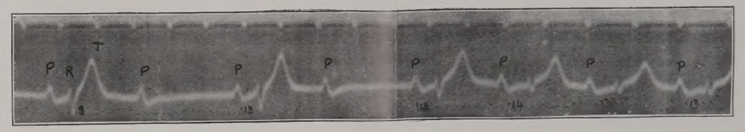

Fig. 4*a*.

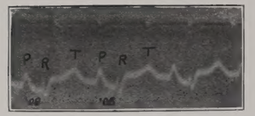

Fig. 4*b*.

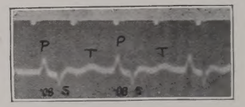

Fig. 4*c*.

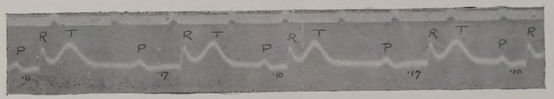

Fig. 5.

regular occurrence. It commences within a few minutes of the cessation of ventilation, and is first manifested by a change of conductivity, a lengthening of the auriculo-ventricular systolic interval. It is observed in the intact curarised animal, with or without vagal section, in the decerebrate and the spinal animal, and after atropinisation.

2. All grades of heart-block, even complete, may be succeeded by speedy and complete recovery of conduction.

3. The heart-block is accompanied by a marked retardation of the auricular rate, and this likewise is independent of inhibitory influences.

4. Alternation of the P-R intervals, in the absence of dropped beats, is described.

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